ARTERIAL HYPERTENSION.

By Arthur R. Elliott, M.D.,
PROFESSOR OF MEDICINE IN THE POSTGRADUATE MEDICAL SCHOOL, CHICAGO.

Abnormally high blood pressure, if once permanently established, is a condition of much significance, as it constitutes a grave departure from the physiological norm and entails certain structural and degenerative secondary effects in the arteries and heart of the most profound character. The mere fact of blood pressure being habitually raised above normal, even to a considerable extent above normal, does not seem necessarily to circumscribe the individual's activities, nor is it always accompanied by noteworthy subjective discomfort. The change to high levels is ordinarily a very gradual affair, the tissues progressively accustom themselves to the new standards of pressure, and the economy may not be disturbed. We consequently often find individuals with systolic readings over 200 mm. Hg. actively engaged in business and professional affairs, unconscious of any disturbance of health. Slowly but surely, nevertheless, the excessive mechanical strain to which their circulatory organs is subjected begets serious degenerative alterations, and finally if the process be not stayed the whole apparatus of the circulation will one day fall to pieces like the "Deacon's one hoss shay." Meanwhile no symptoms of alarming nature may arise until a sudden cardiac failure or an apoplexy tragically reveals the true state of affairs. Recently I was consulted by an active business man who for several weeks had unsuccessfully treated a cough, which was attended by asthmatic symptoms. He was found to have a blood pressure of 245 mm. with a pulse of 124, gallop rhythm, jugular pulse, pulmonary congestion, hepatic hyperemia, and albuminuria. In many a man above sixty we find a basic systolic murmur and accentuated aortic second sound indicating hypertension, or we may encounter indications of a relative mitral leak, with no complaint on the part of the patient beyond a certain puffiness on exertion.

Arterial hypertension, except when it occurs in association with chronic nephritis, is comparatively seldom met with before the age of forty. As an accompaniment of chronic nephritis it is often encountered in early life, even during childhood; otherwise it may be considered a disease of maturity. After forty years is passed the older the patient is when hypertension is diagnosed the more pronounced as a rule will be the element of arterial degeneration and the more apparant the cardiac secondaries. In early middle life unless the elevation of blood pressure is the result of nephritis it is apt to stand unique as the single physical indication of some obscure nutritive or toxic disturbance. In many instances of the kind hypertension is the only thing present without or previous to the development of organic secondaries. Cook advocates the collection of these cases into a separate group for which he suggests the name "essential arterial hypertension." Under this designation he would include that residuum of cases in which after eliminating all the usual and known causes there remains no explanation for the one constant and only physical sign—hypertension of the pulse. In practically all of these cases, as he states, there is a certain element of cardiac hypertrophy, but it may be difficult to recognize. This definition very nearly coincides with the presclerosis of Huchard. I can best describe this type of case by clinical illustration: S. P., aged fifty years, was rejected at a life insurance examination for a policy of large amount, because he had a blood pressure of 165 mm. He consulted me to determine its cause. He was a perfectly healthy looking man of active habits and although a generous liver was not intemperate. He was an excessive smoker. Careful examination failed to reveal any indication of organic disease. His heart may have been hypertrophied, but regarding this, after repeated examinations, I still remained uncertain. The urine was free from albumin and casts and of normal quantity. He complained of flatulency, but was not constipated. His hygiene and diet were carefully regulated, his smoking moderated, and in a year the blood pressure had descended to an average of 135 mm.

This form of primary arterial hypertension is probably more frequent than we are aware, and if the sphygmomanometer were used on all men of middle age coming under observation, both in clinical routine and life insurance examination, attention would be directed to it oftener than is now the case. It is hardly likely that we have to do here with a different type of case from those we ordinarily class as arteriosclerosis with hypertension, or as chronic nephritis. We have encountered it in its incipiency, that is all, before pronounced organic secondaries have developed to stamp the case as arterial, cardiac, or renal. Should the condition persist unmodified we would probably be able to watch the development of arterial fibrosis as the result of prolonged mechanical strain upon the vessels, of cardiac enlargement, casts and albumin, and all the flock of secondaries that characterizes the fully developed disease.

The relationship of arteriosclerosis to high blood pressure still remains difficult to define, notwithstanding the large amount of investigation and discussion devoted to the problem. It is a well known clinical fact that the most extreme degeneration of palpable arteries may exist without elevation of blood pressure above the normal (Dunin, Groedel, Elliott). Equally common in practical experience is the existence of a greatly elevated blood pressure with but slight fibrosis of accessible arteries. We have to account then for the circumstance of an individual with rigid calcareous radials presenting a normal pressure for his age, whereas another patient much younger and possessing vessels far less degenerated gives pressure values double the normal. It would appear that hardening of the superficial vessels alone does not suffice seriously to disturb the normal circulatory pressure. It is necessary that we distinguish very positively between the clinical type of arteriosclerosis and hypertension. They do not by any means go hand in hand and when the two are found together they may only have an indirect relationship. The fact that in most cases of persistent high pressure some fibrosis of accessible arteries can be made out has probably led us to overestimate the importance of arteriosclerosis in elevating blood pressure. Undoubtedly stiff arteries may cause some increase in tension without the operation of any second factor, but that they can unaided produce the excessive values we deal with clinically is open to question. "Arteriosclerosis is an anatomical change, whereas high pressure is a functional disturbance." Instead of arterial changes giving rise to high pressure it is probable that as often as not when the two are found together the sclerosis has been produced by the long-continued strain on the arterial walls caused by the hypertension. As a rule, however, cases of hypertension do not live long enough to develop a high degree of arteriosclerosis. It is clear that some underlying factor not accessible to our present methods of examination must be responsible for the occurrence of high blood pressure in arteriosclerosis. This factor we may assume on general grounds to lie in some disturbance of the splanchnic circulation. In health a certain functional interchange or "give and take" exists between the systemic and splanchnic circuits, the systemic drawing upon the splanchnic at need and at other times using it as a storage for blood.

In disease this "normal balance" may be greatly disturbed and if the splanchnic vessels be the seat of sclerosis their reserve capacity will be reduced and the systemic arterial pressure in consequence raised and maintained above normal. This is practically the conclusion of Hasenfeld and Hirsch, who from clinical and pathological data contend that it is only when the vessels of the splanchnic area or the aorta above the diaphragm are diseased that high pressure develops in arteriosclerosis. For the present at least we may assume that when arterial pressure is persistently raised the terminal divisions of the vascular system (splanchnic and systemic) are principally involved in the sclerosis. This is of the highest practical importance for arteriosclerosis of the splanchnics and arterioles cannot be recognized by physical investigation during life, but may be inferred to exist with a fair degree of certainty from blood pressure observations. The prognosis in this type of case is very different and far graver than in that other order of sclerotics with stiff chalky arteries, but no tension. As Cook has emphasized, the outlook in the case of a robust looking man of fifty-five with a blood pressure of 200 mm., even with no appreciable arterial degeneration, is not so good as in the case of a man of sixty-five with rigid arteries and a pressure of 130 mm. We find many examples of the latter type in every old peoples' home where they live on year after year eventually dying of an atrophy, cerebral or cardiac. The patient with hypertension is in daily danger of apoplexy or heart failure. The element of danger is the tension.

Fraenkel and Hasenfeld have pointed out that corpulent persons of a sedentary habit are prone to develop sclerosis of the splanchnic vessels with high blood pressure. There is much reason to believe that such cases are toxic in origin and it is probable that the chief source of the pressor toxins is the digestive organs. The argument of clinical experience lends weight to the contention of Russel that in non-nephritic cases the hypertension is caused by the presence in the blood of substances which are absorbed from the alimentary tract and are the product in one form or another of what has been swallowed as necessary food or as unnecessary indulgence. This does not imply that the big feeder must necessarily develop splanchnic sclerosis and the small feeder escape. The essental factor will prove to be the digestive and eliminative competency of the individual and the relative suitability of his diet. In summing up the relation between arteriosclerosis and high blood pressure we must acknowledge in the first place that the thickening of the vessel walls incident to age is capable of causing a gradual, but by no means extreme rise in the average arterial pressure. We see this in the slow increase of average pressure readings as life advances, a pressure of 140 mm. being accounted normal for a man of sixty-five years, whereas his son will have a pressure of 120 mm. and his grandson 100 mm. Should the pressure of such a man register persistently above 160 mm. and with this his heart show enlargement he may be regarded as having hypertension and we must invoke some cause other than his arteriosclerosis to explain it. Such a development means that in addition to a thickened artery he has a constricted one, the former an anatomical change, the latter a spastic condition produced by some toxic excitant circulating in the blood and causing hypertonic contraction of the arterioles, splanchnic or systemic (Russel).

It is hardly necessary to urge the importance of carefully investigating the condition of the urine in every instance of hypertension. The frequency with which chronic renal disease is associated with cardiovascular changes is well known and no factor is so potent as nephritis in the production of high blood pressure. If a diagnosis of chronic interstitial nephritis can be made it is not necessary to search farther for the cause of high pressure. At the same time it is to be remembered that greater care than ordinary is required to diagnose nephritis in the presence of arteriosclerosis with high blood pressure, owing to the fact that some degree of atrophy of the kidneys, manifested by slight albuminuria and casts, is almost always present as a consequence of those organs sharing in the general vascular deterioration. Moreover, almost all cases, no matter of what origin, showing blood pressures over 200 mm. will display some albumin in the urine. It is unfortunate that no clear distinction is made in clinical literature between arteriosclerotic renal atrophy, and true contracting kidney. Every experienced clinician knows how vast the difference is in course, prognosis, and treatment between the two conditions, and yet it is only from experience and not from medical literature that one learns to appreciate the distinction. Chronic interstitial nephritis is an inveterate organic lesion showing severe toxic manifestations, a steady and even rapid downward progression, is not amenable to treatment, and has a bad prognosis. In every respect, no matter how similar may be its urinary and physical indications, arteriosclerotic renal atrophy is the opposite of this, being slow in development and progress as is the case with the sclerotic atrophies generally. A distinction between the two is important owing to the difference in prognosis.

Chronic interstitial nephritis gives rise to the highest systolic readings that are observed clinically, pressures of 300 mm. and higher being recorded. The highest record I have is 285 mm. in a young woman, who died a fortnight after in uremic coma. In a study of 60 cases of chronic nephritis[1] the average pressure was 190 mm. Chronic nephritis is essentially a disease of systemic scope, involving the heart and arteries as well as the kidneys. Arterial hypertension is one of its salient features. Notwithstanding occasional exceptions to the rule, high pressure is so significant that it constitutes one of the most valuable diagnostic indications of that disease. The use of the sphygmomanometer and the discovery of high pressure will at once put the observer on the alert.

The attempt to establish a working hypothesis to account for all the varieties of hypertension leads us face to face with the toxic theory. Arterial hypertension is best exemplified as it occurs in a group of chronic diseases having as their common essential characteristic, toxemia. In all probability the *materies morbi* consists of certain abnormal biochemical products present in the circulating blood. This is apparent in scarlatinal nephritis in which the tension rises a few hours after the appearance of albumin in the urine, entirely too early for the rise to be explained by the formation of arterial fibrosis. The high tension of uremia is another instance in point. For laboratory proofs we have the well-known fact that the pressor principle of adrenal and pituitary glands and also certain drugs (ergot, nicotin, digitalis) will raise blood pressure. That hypertension is a functional effect, as well as an organic product, we may infer from the clinical observation that measures designed to detoxicate the system (diet, sweats, cathartics) will result in some reduction of pressure in most cases of hypertension, and the therapeutic action of the nitrites could not be secured did the condition rest solely on a basis of structural change.

---

[1] Jour. Amer. Med. Assoc., April 13, 1907

The end-effects of long continued high blood pressure are manifested principally in the heart and arteries. The arterial walls reacting to the excessive mechanical strain undergo a progressive structural deterioration to the great prejudice of their normal histology and vasomotor tonus. In the end vasomotor response may be so seriously disturbed that nitrites may fail to lower blood pressure. Peripheral resistence increases in this manner with the stage of the disease. A somewhat parallel sequence of events is apparent in the heart. At first in response to overwork the myocardium hypertrophies just as does the myarterium. The peripheral retard being persistent and increasing and the heart reserve limited, myocardial insufficiency becomes inevitable. The heart at first hypertrophies and then dilates in the face of continued overstrain.

The complications of arterial hypertension will be determined by the ability of the different organs to withstand the strain. On the part of the heart we observe hyposystole and asystole, of the arteries atheroma, of the kidneys albuminuria and uremia. A cerebral vessel may give way and apoplexy close the scene.

The symptomatology of arterial hypertension is general rather than special. There may be an entire lack of symptoms until vertigo or an attack of acute dyspnœa alarms the patient, or some cerebral accident occurs. The condition is often revealed quite unexpectedly during examination for life insurance. Frequently the earliest symptoms are of nervous type, irritability, depression of spirits, disturbed sleep, or it may be that the patient complains of bilious symptoms, flatulency, constipation, headaches, and vertigo. There is usually precordial discomfort and dyspnœa following effort and the patient rises once or more at night time to void urine. As a rule the night urine exceeds in quantity that passed during the day. A symptom noted in a number of my cases is a severe paroxysmal flatulency nocturnal in occurrence or developing on exertion. During the intervals between attacks there may be no complaint of flatulency, and examination at any time may fail to reveal special tympany or distention. Shortly after retiring for the night the patient may experience a feeling of distention and oppression across the lower chest or he may awake after a period of sound sleep with a feeling as if the stomach were full of gas. Instinctively he strives to relieve himself by gulping, drinking hot aromatic or alcoholic drinks, and the fact that comfort is reëstablished, frequently after an hour or more, only when he has belched freely, confirms him in his idea that it is indigestion. The breathing is usually hurried, and palpitation may coincide. Attacks of this nature may develop after exertion, especially soon after meals and most frequently during the early part of the day. Bending over, lifting, straining at stool may precipitate the symptom. This development is probably similar in character and significance to cardiac asthma and denotes insufficiency of the right heart. All of the patients with this symptom whom I have seen, have had enlarged livers and one a well marked jugular pulse. I have come to regard flatulency of irregular and paroxysmal occurrence in mature individuals as extremely significant, and I believe that every such case should be carefully investigated as to the cardiovascular condition. A typical instance of this character is the following case.

S. F., aged seventy-six years, and weighing one-hundred and ninety pounds, is a retired merchant of means, and boasts that he has never been sick in his life. He confesses that he has been a very hearty eater and heavy smoker, and has for years been constipated. Although rather spare in his limbs he is round-bellied and his face is ruddy and somewhat pigmented. He complains that for some months he has been greatly troubled with attacks of explosive belching of gas developing during exertion and interfering greatly with his activities. These attacks are frequent during the early part of the day, especially after breakfast and occur comparatively seldom and less severely toward evening. They come on during walking, especially if the weather is cold or it is windy or the walking rough. He is compelled to sit down or lean against a fence or building until he had relieved himself by belching. There is little intestinal flatus, and but slight dyspnœa between the attacks. He is somewhat puffy on exertion. He rises two to four times at night to void urine. He is found on examination to have a greatly enlarged heart, the apex 17 cm. from the midsternum; and a systolic blow at the mitral area reveals the lea of dilatation. The blood pressure ranges from 190 mm. to 225 mm. There are no urinary indications of nephritis. This patient remained under observation for two months with slight benefit to his symptoms and then departed south to a more agreeable winter climate. Two weeks after his departure he died of acute heart failure.

It is noted frequently by patients with hypertension that smoking causes restlessness and cerebral discomfort and that heavy meals are not so well borne as formerly. Women are apt to complain of flushing and burning of the face, and if in the middle period of life are apt to ascribe it to the climacteric. I have noted as a prominent symptom in two cases pains of an anginoid character referred to the precordium, the left arm, or abdomen following exertion. Tachycardia and palpitation are complained of. As a sign of great significance and rather grave import, as it indicates the beginning of hyposystole, is dyspnœa on lying down. The explanation of this development lies in the fact that the arterioles being contracted the blood collects unduly in the veins especially the splanchnic veins. When the patient is upright these veins act as a reservoir, but when he lies down the force of gravity tends to empty them into the right heart. This leads to overdistention of the pulmonary capillaries and dyspnœa. The only way rightly to interpret these symptoms is to take the blood pressure. With every individual of middle age complaining of persistent functional disturbance this precaution should never be omitted.

Examination of the heart in cases of hypertension will reveal enlargement of that organ. This may sometimes be difficult of detection in fat individuals and in women with pendant breasts. In the earlier stages the heart sounds are usually clear, the first tone booming and prolonged, the aortic second loud, valvular, and ringing. If the ventricle has dilated the murmur of relative mitral insufficiency may be heard. Late in the case with the heart badly disorganized, the patient dropsical, and the pulse small and arrhythmic, it may become extremely difficult to determine whether the case is one of cardiac failure from prolonged hypertension or a valve lesion in the final stage of incompensation. The sphygmomanometer may afford us no help at this juncture, owing to the failure of the ventricle having so impaired the support of the circulation as to induce secondary low blood pressure. One must then fall back upon the history and the general features of the case to decide the point.

Analysis of the urine in arterial hypertension may reveal no morbid elements from the kidney, although in cases of some standing a few hyaline casts will usually be found. If chronic nephritis exists the usual urinary characteristics of that disease will appear to point to the origin of the hypertension. Renal permeability to albumin seems to be overcome when the blood pressure reaches or exceeds 200 mm., so that cases with very high pressures, whether primarily nephritis or not, usually have albumin in the urine.

THE USE AND ABUSE OF GASTRO-ENTEROSTOMY.[1]

By John B. Deaver, M.D., LL.D.,
SURGEON-IN-CHIEF TO THE GERMAN HOSPITAL, PHILADELPHIA.

Gastro-enterostomy, "the keystone of gastric surgery," was first performed in 1881. Today this operation is being frequently made. The object of this paper is to excite discussion upon the bearing of the operation along the lines where it is proper as well as improper. The risks of gastro-enterostomy performed by an experienced surgeon are inconsiderable. The mortality of gastro-enterostomy in benign disease is low, 1 to 2.5 per cent. This is fortunate in a sense, and yet unfortunate if it encourages the performance of the operation in conditions in which it is not properly indicated.

A most important, essential, and interesting point is that metab-

---

[1] Read at a meeting of the Manhattan Medical Society, New York, December 17, 1909.